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Huntington's like conditions in China, A review of published Chinese cases

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Abstract

Background: Knowledge about HD in China is lacking in the international literature. We have therefore analyzed the Chinese literature to thoroughly explore the clinical characteristics of Huntington disease in China.

Methods: A computer-based online search of China National Knowledge Infrastructure was performed to review case reports concerning HD published between January 1980 and April of 2011, and the clinical characteristics were extracted.

Results: A total of 92 studies involving 279 patients (157 males and 122 females) were collected, 82.0% of which were from provinces of North China. Most of the cases (97.8%) had a family history of HD, and paternal inheritance (65.5%) was higher than maternal inheritance (34.5%). Onset age was 35.8 (± 11.8) years, death occurred with 45.6 (± 13.5) years after a course of 11.6 (± 5.6) years. Involuntary movements were the most frequent reported presentation (found in 52.3%, including 64.4% in the entire body, 19.8% in the upper limbs, and 13.7% in the head and face). Psychiatric symptoms at onset were reported in 16.1%, and cognitive impairment in 1.8%. With disease progression, 99.6% of patients had abnormal movements, 67.9% cognitive impairment, and 35.0% suffered psychiatric symptoms. Of the reported patients, only 22 underwent IT15 gene testing with positive results.

Conclusion: HD is a well-reported entity in Chinese medical literature, however, only a small number of instances have been proven by molecular diagnosis. Most of the features resemble what is known in other countries. The highly predominant motor presentation, and the higher male prevalence as well as the apparent concentration in Northern China may be due to observational bias. There is therefore a need to prospectively examine cohorts of patients with appropriate comprehensive assessment tools including genetic testing.

Introduction

Huntington disease (HD) has been mainly reported in the West and, to date, knowledge on HD in China is very sparse. It has been claimed, that the incidence is very low, however, well-conducted epidemiological studies are lacking. Furthermore, the phenotype in HD patients has not been well characterized, and it may well be that it is influenced by ethnic background. We have therefore performed an analysis of the literature on HD China by systematically retrieving appropriate reports to describe the clinical manifestations of the disease in this country.

Methods

Items including Huntington's disease, Chinese equivalents of the terms: hereditary chorea, chronic progressive chorea, and hysterical chorea were used as keywords to search articles published in the China National Knowledge Infrastructure. Pre-indexing did not reveal any systematic evaluation, prospective or retrospective cohort study regarding HD in China. Case series and individual reports published between January 1st , 1980 and April 30 th , 2011 were included, but reviews and experimental studies on HD excluded. Case reports with non-definite clinical diagnosis or repetitive contents were excluded. Information was extracted about family history, involuntary movements, including the region involved, cognitive disturbances, and psychiatric symptoms. Results of imaging and neurophysiological studies, and of genetic testing were included, where available.

Literature inclusion

A total of 230 articles related to HD were collected; 136 were excluded since they reported identical data and basic studies; 94 studies involving 547 patients were selected (Table 1). Of these, 306 cases ad to be excluded due to incomplete clinical data. In total, 279 patients were included (157 males and 122 females), with an age of onset of 6-70 years. Of these cases, 236 cases (96.8%) included a family history. A total of 89 patients were noted to have come from a precise region, 16 were from the South, and the remainder were from the North (82.0%), including 15 from Henan Province, 14 from Shandong Province, and 8 from Hebei Province.

Table 1. Reports included in the present survey

Year of publication	First author	Male		Female		Molecular testing	Ref.
		N	Age at onset	N	Age at onset		
1980	Wancong Gao	0	na	1	na	na	[11]
1980	Linde Liu	1	42	1	27	na	[12]
1980	Wenshi Li	2	37 to 42	1	54	na	[13]
1980	Xiongya Wu	1	51	1	47	na	[14]
1983	Qian Xu	5	15 to 44	3	7 to 8	na	[15]
1984	Zian Chen	4	32 to 39	1	32	na	[16]
1985	Xiaolian Du	2	5 to 40	3	16 to 19	na	[17]
1985	Jixue Feng	0	na	1	41	na	[18]
1985	Shuyou Fang	4	28 to 46	1	24	na	[19]
1986	Guiqing Wang	2	28 to 58	1	28 to 58	na	[20]
1986	Honglin Fu	0	na	3	34	na	[21]
1986	Hua Shao	1	39	0	na	na	[22]
1987	Wenjun Chen	1	31	0	na	na	[23]
1988	Shicheng Pei	1	43	0	na	na	[24]
1988	Shuqiang Bu	0	na	2	34 to 37	na	[25]
1988	Keqing Ding	3	41 to 55	1	45	na	[26]
1988	Zhiyuan Ha	1	28	1	30	na	[27]
1989	Yongqian Xing	5	20 to 35	3	25 to 43	na	[28]

1989	Xuesong Tu	0	na	1	na	na	[29]
1989	Fuyuan Shao	1	7	0	na	na	[30]
1990	Yuxiang Xu	4	17 to 42	1	35	na	[31]
1990	Chuandong Wu	2	17 to 36	0	na	na	[32]
1990	Jiaxi Huang	0	na	4	34 to 40	na	[33]
1991	Zhaoxiang Zeng	0	na	1	31	na	[34]
1992	Ke Fan	4	27 to 38	2	38 to 56	na	[35]
1992	Changdao Sun	3	24 to 30	2	21 to 25	na	[36]
1992	Chengyu Chen	4	27 to 47	0	na	na	[37]
1993	Fengying Hou	1	47	0	na	na	[38]
1994	Yanjun Gao	1	48	0	na	na	[39]
1994	Qishan Dong	1	42	1	38	na	[40]
1994	Yuejin Huang	0	na	1	42	na	[41]
1995	Xiaomei Yao	0	na	2	46 to 47	na	[42]
1995	Xiuhua Fan	1	47	0	na	na	[43]
1995	Chenghao Chu	4	10 to 65	1	20	na	[44]
1995	Jiying Wang	0	na	1	32	na	[45]
1995	Ronghua Yong	1	60	1	48	na	[46]
1996	Kun Liu	0	na	1	42	na	[47]
1996	Zhaozhong Shen	2	na	0	na	na	[48]
1997	Meiju Hou	1	36	0	na	na	[49]
1997	Jiaming Xia	3	18 to 35	2	18	na	[50]
1997	Wenhua Sun	0	na	1	25	na	[51]
1998	Xiangming Fang	5	18 to 40	0	na	na	[52]
1998	Weizhou Liu	1	23	0	na	na	[53]
1998	Yujin Zhang	6	24 to 50	4	30 to 55	na	[54]
1998	Xiaoping Yang	2	30 to 40	2	13 to 33	na	[55]
1998	Kai Feng	1	56	0	na	na	[56]
1998	Zuozhi Gao	2	33 to 54	1	34	na	[57]
1998	Xiaoping Zeng	3	43 to 54	1	45	na	[58]
1998	Yuchen Sun	1	42	2	45 to 46	na	[59]
1999	Weiguo Yang	0	na	1	39	na	[60]
1999	Guilan Wang	0	na	1	8	na	[61]
1999	Bin Liu	4	32 to 45	3	34 to 38	na	[62]
2001	Xi Zhang	1	50	0	na	na	[63]
2001	Jing Chen	1	24	0	na	na	[64]
2001	Wen Li	2	21 to 26	3	25 to 26	na	[65]
2001	Qinglin Dong	4	23 to 36	2	30 to 32	na	[66]

2001	Huizhi Fan	0	na	1	32	na	[67]
2001	Liansheng Xu	5	29 to 54	5	32 5o 52	na	[68]
2002	Jing Liu	3	28 to 46	5	35 to 50	na	[69]
2002	Benqiang Deng	1	66	0	na	na	[70]
2002	Ye Tian	1	70	1	40	na	[71]
2003	Liqun Fang	1	31	0	na	na	[72]
2003	Huize Ma	2	31 to 52	3	33 to 45	na	[73]
2003	Yuhai Zhao	2	22 to 40	1	30	N CAG repeats	[74]
2004	Feng Tian	1	54	2	38 to 50	na	[75]
2004	Mingbing Chen	1	32	0	na	na	[76]
2004	Weiwei Dong	1	14	0	na	na	[1]
2004	Jun Chen	2	52	2	44 to 54	na	[76]
2004	Jiamu Wu	2	31 to 33	0	na	na	[77]
2004	Beilei Zhu	1	28	0	na	Genetic test (no N CAG repeats)	[78]
2005	Liyan Guo	0	na	1	32	na	[79]
2006	Shiyong Zhang	0	na	1	52	na	[80]
2006	Huamei Wang	2	41 to 48	0	na	na	[81]
2006	Fang Lin	4	421 to 58	0	na	na	[82]
2006	Baorong Zhang	3	30 to 45	5	18 to 36	N CAG repeat	[83]
2006	Zhilin Shi	2	33 to 45	1	45	Genetic test (no N CAG repeats)	[84]
2007	Yuan Liu	3	33	1	17	N CAG repeats	[85]
2007	Yanchun Geng	2	28 to 29	0	na	na	[86]
2008	Wei Xu	3	25 to 36	2	27 to 45	Genetic test (no N CAG repeats)	[87]
2008	Jin Yu	1	41	4	32 to 45	na	[88]
2008	Ning Wang	1	na	2	na	N CAG repeat	[89]
2008	Zhouri Li	2	40 to 43	1	41	na	[90]
2008	Yiming Feng	1	33	0	na	na	[91]
2009	Xingwang Song	2	41 to 57	4	6 to 58	N CAG repeat	[92]
2009	Qiuhong Zheng	2	50 to 60	3	41 to 60	na	[93]
2009	Meiying Cai	1	30	2	35 to 45	N CAG repeat	[94]
2009	Weijie Liu	0	na	1	37	na	[95]
2010	Meihua Zhu	0	na	1	35	na	[96]
2010	Ge Gao	3	36 to 42	0	na	N CAG repeat	[97]
2010	Jing Ma	0	na	1	46	na	[98]
2011	Min Li	0	na	1	51	na	[99]

	Total	157	122		

Most (65%) of the patients had onset in middle age (Table2), with a mean of 35.8 years (± 11.8), mean age at death was 45.6 years (± 13.5, range 13–69), and mean course from onset to death was of 11.6 years (± 5.6, range 3–30). Around 9 % had a juvenile onset. The study included 115 families. Paternal inheritance was more often found than maternal inheritance (Table 2), and age of onset with paternal inheritance was lower than maternal inheritance (34 ± 10 versus 37 ± 10 years, $P < 0.05$). The age at death (46 ± 15 vs. 50 ± 10 years), and the course of disease (12 ± 6 vs. 12 ± 4 years), were not significantly different. In addition, no difference was found between male and female patients in terms of age of onset, death age (47 ± 14 versus 46 ± 12 years), and course of disease (12 ± 6.0 vs. 10.7 ± 5.0 years). Within the cohort, 55 patients death were reported, of which three were due to suicide.

Table 2. Clinical features of the reported patients

	N of total with available data	%
Inheritance		
Paternal	127/194	65.5
Maternal	67/194	34.5
Anticipation	42/59	71.2
Age at onset (yr)		
< 20	24/279	8.6
30–55	182/279	65.2
Presentation at onset		
Abnormal movements	146/196	52.3
Generalized	94/146	64.4
Head and face	20/146	13.7
Upper limbs	29/146	19.8
Lower limbs	3/146	2.1
Psychiatric disorder	45/196	16.1
Cognitive impairment	5/196	1.8
Course		
Abnormal movements	242/243	99.6
Psychiatric disorder	85/243	35.0
Cognitive impairment	165/243	67.9

The most frequent presentation at onset were abnormal movement found in more than half of the patients (Table 2), with a predilection for the face and upper limbs when it was not generalized. In the course of the disorder most patients developed abnormal movements, followed by psychiatric symptoms and cognitive impairment (Table 2). There were some specific features in single patients, for example, one displayed speech impairment, instability of gait and cognitive impairment, with no obvious involuntary movement[1]. Dysarthria and dysphagia was also reported during the course in 26 % of the patients. One single patient had epilepsy at onset.

Laboratory investigations

In a total of 48 patients with electroencephalograms, 34 (70.8%) had abnormal curves, mostly with mild slowing. In a total of 16 patients undergoing cerebrospinal fluid examination, three (18.8%) displayed abnormally increased protein levels. In a total of 89 patients undergoing cranial imaging, 80 (90.0%) presented with abnormalities of varying extent, including 65 (73.0%) with brain atrophy and lateral ventriculomegaly, 2 (2.2%) with selective caudate nucleus atrophy, and 26 (29.2%) with brain atrophy, lateral ventriculomegaly, and caudate nucleus atrophy. IT15 gene detection was reported positive in 38 patients, of which CAG repeats were clearly reported in 33 of the cases. The number of CAG repeats was greater in patients with a younger age of onset (mean of 61 in patients with onset before versus 46 with age of onset after 30).

Discussion

The prevalence of HD is quite variable, with figures varying between 0.5 (Finland)[2], 1 (Croatia [3]) and 10

(German speaking European countries [4]) per 100 000 in Europe, and with high local prevalence in some communities, like in Venezuela (almost 700 in the Lake Maracaibo region [5]. It is usually thought that the prevalence in Asia is lower, however, fewer data than in the West have been reported so far. In Japan reported estimations ranged between 0.1 [6] and 0.7 [7]. Earlier estimation in Hongkong are within the same range [8], however, no data have been so far reported for mainland China. The present survey of Chinese literature on HD shows that the disease is indeed present in this country, but does not provide precise clues on the prevalence of the disorder. It also suggests a higher prevalence in Northern China, however this may also be a report bias. The number of juvenile cases reported seems higher than in other regions of the world and there is a male predominance in overall prevalence. However, the mean age of onset is otherwise consistent, but the course seems shorter with absence of the gender difference reported earlier [9]. The majority of patients had a positive family history, and only five cases were determined to be sporadic. Moreover, the number of cases due to paternal inheritance was significantly greater than that from maternal inheritance, with a significantly younger age of onset with paternal inheritance, which was in accordance with previously published results. Only one case among the 20 with juvenile onset was reported to have epileptic seizure, which is in contrast to the literature, reporting up to 30% of them. Studies have suggested that the suicide rate of HD patients is significantly greater than healthy individuals, in particular in early or advanced stages [10]. Only three patients were reported to have committed suicide, however, data regarding suicide in China are not available for comparison. In general the other aspects were similar to the reports in other populations. However, only a small number had a molecular confirmed diagnosis, but the trend to earlier age of onset with higher triplet repeat numbers is found here also.

In conclusion, ethnic differences among Chinese with HD as compared to other populations are possible. However, the use of appropriate clinical assessment tools and molecular genetic testing in a larger cohort of patients is urgently needed. For this reason a Chinese Huntington’s disease network is going to be launched.

Competing interests

The authors have declared that no competing interests exist

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